Protective Mechanisms of Plant Based Antioxidants Against Pesticide Induced Cellular Toxicity

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Abstract-

Oxidative stress, mitochondrial dysfunction and DNA damage caused by exposure to pesticides result in organ toxicity and contribute to the development of diseases. The antioxidants present in plants, which include polyphenols, flavonoids, carotenoids and vitamins, protect against these damaging effects through different pathways. These include scavenging reactive oxygen species, metal chelation and induction of antioxidant enzymes like superoxide dismutase, catalase and glutathione peroxidase. Moreover, antioxidants also modulate inflammatory pathways, inhibit apoptosis and prevent mitochondrial damage. It has been shown in preclinical studies that Curcuma longa, Camellia sinensis and Moringa oleifera protect against pesticide induced liver damage, neurological damage and DNA damage.

Introduction-

Pesticides are extensively used in agriculture to increase production, but they pose a great risk to health owing to the excessive use of pesticides. The accumulation of the residue of these pesticides leads to oxidative stress through the creation of reactive oxygen species, thus causing cellular toxicity and DNA damage. Prolonged use of pesticides causes neurotoxicity, hepatotoxicity, immunotoxicity and cancerous growth. Due to their properties that allow for antioxidant activity, plant derived antioxidants have been gaining popularity as they counter oxidative stress. Medicinal plants contain polyphenolic compounds, flavonoids and terpenoids among others.

Materials and Methods-

1. Literature Search and Data Collection-

A systematic search was done in PubMed, Scopus, Web of Science and ScienceDirect for published literature from 2000-2026 with keywords including “plant antioxidants”, “pesticide toxicity”, “oxidative stress”, “cellular protection”, “flavonoids" and “polyphenols”. Boolean operators (AND/OR) were used for more relevant literature. Articles included were peer-reviewed studies with in-vitro and in-vivo investigations. Those excluded were articles not having mechanistic information, not in English language and abstracts of presentations from conferences. A total of 182 references were found with 94 being finally analyzed.

2. Selection of Pesticides and Plant Antioxidants-

Pesticides causing oxidative toxicity commonly in use include organophosphates like chlorpyrifos and malathion, organochlorines like DDT and lindane, carbamates such as carbaryl and pyrethroids like cypermethrin. Plant antioxidants were further classified into four categories: polyphenols from plants of Camellia sinensis, Vitis vinifera and Curcuma longa; flavonoids from Moringa oleifera, Ginkgo biloba and Allium sativum; carotenoids from Daucus carota and Solanum lycopersicum and vitamins C and E from Emblica officinalis and Triticum aestivum. The dosage and duration of treatment for each compound was compiled from different literatures.

3. Toxicity Test Experimental Models -

The data obtained from three experimental models were collected. The in vitro models had human hepatoma HepG2 cells, SH-SY5Y neuronal cells and primary rat hepatocytes that were exposed to pesticides at an IC50 concentration. Determination of cell viability was performed using the MTT, LDH leakage and trypan blue methods. In vivo model studies involved Wistar rats, Swiss albino mice and zebrafish which were treated with pesticides orally or intraperitoneally for periods ranging from 7 to 90 days. The organs studied included the liver, brain, kidney and testis.

4. Biochemical and Molecular Assays-

The biomarkers of oxidative stress determined include reactive oxygen species (using DCFH-DA), malondialdehyde as marker of lipid peroxidation and 8-OHdG as marker of DNA oxidation. Levels of antioxidant defense were determined through SOD, CAT, GPx, GSH-R activity and levels of reduced glutathione. For evaluation of mitochondrial function, measurements of mitochondrial membrane potential through JC-1 dye staining, ATP levels and enzymatic activities of

complexes I and III were performed. Determination of apoptosis and inflammation included measurement of caspase-3 and -9 activities, Bax/Bcl-2 ratio, levels of TNF-α, IL-6 and NF-κB using ELISA and Western Blot, respectively.

5. Preparation and Standardization of Plant Extracts-

The techniques employed for extraction of plant extracts have been reported. They involve aqueous, ethanolic, methanolic and hydroalcoholic extractions, with or without the use of ultrasound or microwaves. The standardization of phytochemicals involved determination of the total content of phenolics using the Folin-Ciocalteu procedure, total content of flavonoids using the aluminum chloride method and high performance liquid chromatography analysis of the major components like curcumin, epigallocatechin gallate, quercetin and lycopene.

6. Data Analysis and Interpretation-

The quantitative data were analyzed and transformed into % protection compared to the "pesticide only" treatment groups. The protective action was categorized into direct antioxidants, metal chelation, enzyme induction, anti-inflammatory, anti-apoptosis and mitochondrial stabilization. Significance of findings at $p<0.05$ as stated in original literature was also taken note of. Dose response and time response trends were documented.

Results and Discussion-

The antioxidants derived from plants effectively mitigated oxidative stress caused by pesticides in both in vitro and in vivo systems. The polyphenolic and flavonoid molecules lowered ROS, MDA and protein carbonyl production, while simultaneously increasing the activities of SOD, CAT and GSH. The extracts of Curcuma longa and Camellia sinensis conserved mitochondrial membrane integrity, lowering the caspase-3 activation. On a mechanistic note, the antioxidants stimulated the expression of the Nrf2/HO-1 pathway, prevented the activation of the NF-κB signaling responsible for inflammation and bound to metal ions that are involved in pesticide mediated toxicity. The protective effects of quercetin and EGCG were evident when tested against chlorpyrifos and cypermethrin in HepG2 and neuronal cells.

References-

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